factors is in general surprisingly low when the financial[8] and emotional costs[9] to the patient of attending for services that are sometimes of questionable value are considered.[10] Other studies support the suggestion that aspects of the service may offer explanations for non-attendance. A study of abortive ambulance journeys found that half of wasted journeys could be attributed to administrative errors such as not notifying the patient or sending the ambulance when the appointment had been cancelled or when the patient had already been admitted.[11] A Canadian study reported that an appreciable proportion of non-attendance could be attributed to the poor communication of appointments.[12]

The distinction between those factors relevant to non-attendance that arise within the patient population and those that stem primarily from the organisation of the service is important for management. Though there may be interaction between factors arising from patients and from hospital organisation, their implications for planning improvements are quite different. If non-attendance were primarily a reflection of patients' generalised indifference to the service offered then the problem would not be amenable to any straightforward remedies. The identification of important factors of communication and administration, however, suggests that improvements can be expected to follow from fairly simple adjustments in procedure.

Meticulous organisation may produce remarkably low non-attendance rates.[13] Intervention trials in Canada and the United States have shown that telephone and postal reminders can greatly reduce non-attendance rates.[11 14] Before attributing the problem of non-attendance in the United Kingdom to "the need for hospital patients to recognise their moral responsibility to turn up on time for appointments in order not to waste valuable NHS resources"[1] it is important to ensure that administrative arrangements are likely to facilitate attendance.

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First myocardial infarctions in Asian and white men

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Abstract

***Objective*—To compare the presentation and natural course of first myocardial infarctions in immigrant Asians and the indigenous white population in Britain and the subsequent risk states of the two groups.**

***Design*—Prospective ethnic comparison of consecutive patients with first myocardial infarctions.**

***Setting*—Secondary referrals to a coronary care unit of a district general hospital.**

***Patients*—128 Men (77 white, 54 Asian) presenting consecutively with a first myocardial infarction diagnosed on the basis of clinical, biochemical, and electrocardiographic findings.**

***End point*—Identification of mechanisms accounting for the increased rate of ischaemic heart disease in Asians.**

***Measurements and main results*—Infarct size was assessed by measuring the release of creatine phosphokinase (all patients), radionuclide ventriculography (50), and contrast ventriculography (103). Risk states after infarction were assessed from the degree of ventricular dysfunction as determined by exercise electrocardiography (82 patients) and from the extent of coronary atheroma as determined by coronary arteriography (103). Glucose state was measured in fasting venous blood samples. Overall the relative rate of infarction was 4·9 times higher in Asians (95% confidence interval 3·4 to 6·9) than in the white population. Moreover, the relative rate of infarction was higher in Asians in all 10 year age groups, the greatest difference being in 30-39 year olds. The mean age of the Asian denominator population was 47·1 years compared with 49·5 years in the white population. Age at infarction was less in Asians (50·2 years) than in white patients (55·5 years; mean difference 5·5 years (95% confidence interval 2·5 to 7·1)). In Asians the mean creatine phosphokinase activity was 777 (95% confidence interval 155 to 1399) U/l higher, radionuclide ejection fraction 8·9% (1·0% to 16·9%) lower, and left ventricular fractional shortening 4·8% (1·4% to 8·2%) lower than in white patients. The extent of coronary atheroma was significantly greater in Asians. The mean numbers of plaques in vessels not associated with infarction were 3·66 (median 3·0, range 0-10) in Asians compared with 1·97 (median 2·0, range 0-6) in white patients ($p<0·001$), and a higher proportion of Asians had three vessel coronary artery disease ($p<0·001$). Asians with diabetes or impaired glucose tolerance did not differ from those with normal blood glucose values.**

***Conclusions*—Atherogenesis arises earlier in Asians, contributing to premature first myocardial infarctions. The increased incidence of diabetes in Asians may not in itself be relevant in the greater propensity to coronary atheroma in Asians.**

Introduction

The morbidity and mortality from ischaemic heart disease have been shown to be significantly higher in immigrant southern Asians living in the United Kingdom than in the indigenous population.[1-3] Retrospective analysis of hospital records suggests a more than twofold greater incidence of acute infarction,[4] and

a small study in London[5] and a prospective study in Birmingham (paper delivered to the British Heart Foundation workshop on "Asians and coronary heart disease," London, January 1988) found infarction rates three to five times higher in Asians. The increased risk of ischaemic heart disease seems to apply to all immigrant southern Asians irrespective of their religion, dietary habits, or socioeconomic state.[6]

A parallel finding in all immigrant Asian communities is the high incidence of diabetes mellitus and impaired glucose tolerance.[7,8] Given the known association between diabetes and ischaemic heart disease[9] some workers have tried to explain the high incidence of ischaemic heart disease in Asians on this basis.[5,10] The role of diabetes in the aetiology of ischaemic heart disease in Asians, however, remains unclear.

This study was undertaken to compare the relative rate, clinical presentation, and progression of acute first myocardial infarctions in Asians and white people living in Britain. In particular the comparative severity of coronary atheroma between ethnic groups and between normoglycaemic Asians and those with abnormal glucose tolerance was investigated.

Subjects and methods

Over two years from June 1985 to June 1987, 128 men presenting consecutively with a first myocardial infarction were recruited into the study. Entry was restricted to those aged below 60 and resident in the London borough of Harrow at the 1981 census. This facilitated an estimation of relative rates of infarction by using data from the census to estimate the denominator population for each ethnic group. Men with a previous history of myocardial infarction or with electrocardiographic evidence of previous infarction remote from the site of current infarction were excluded.[11]

Patients were classified as white if three out of four grandparents had originated from the United Kingdom, Ireland, or Europe and as Asian if three out of four grandparents had originated from India, Pakistan, or Bangladesh or had migrated to Britain from east Africa.

Possible bias in referrals was investigated by a postal survey of 99 local general practitioners. They were asked if in cases of suspected myocardial infarction they had different thresholds of referral for different ethnic groups and whether they preferentially referred patients to Northwick Park Hospital as opposed to other hospitals in the area. Eighty three replies were received and no ethnic bias in terms of threshold of referral or preferential referral to Northwick Park Hospital was found. On average, practices reported that 20% (range 5-40%) of their lists were ethnic Asians.

Myocardial infarction was diagnosed in the presence of any two of the following: a clinical history compatible with infarction; a serial rise and fall in creatine phosphokinase activity (or in the absence of a total rise above 300 U/l a creatine phosphokinase MB fraction of >10%); and typical electrocardiographic changes persisting for a minimum of 24 hours. Patients were designated as having had a non-Q wave, anterior Q wave, or inferior Q wave infarction according to established criteria.[12,13]

Creatine phosphokinase estimations were made on admission, then every 12 hours for the first 36 hours. Thereafter samples were taken daily for a further two days. The character and site of pain were assessed by two independent members of the medical staff in the patient's first language. Pain was scored for site and character as: crushing, squeezing, or pressing—score 0; sharp, pin-like stabbing—score 1; other or no pain—score 2; central or retrosternal—score 0; left precordial—score 1; other—score 2. All patients with total scores of ≥ 1 were classified as having atypical pain. Before discharge patients were classified as normoglycaemic, having impaired glucose tolerance, or diabetic on the basis of a fasting blood glucose estimation according to World Health Organisation criteria.[14] Six weeks after infarction exercise electrocardiography was performed in all survivors except those with unstable angina, clinical heart failure, left bundle branch block, or permanent pacemakers and those who had received coronary artery bypass surgery or balloon angioplasty since their infarction. Exercise was performed in a temperature controlled laboratory as described.[15]

Rest and exercise radionuclide ventriculography was performed on the first 25 patients in each ethnic group six to eight weeks after discharge. Data were analysed by two independent observers who were unaware of the patient's name and ethnic origin.[16,17] Myocardial perfusion scintigraphy was performed on the first 40 patients entering the study. Exercise and redistribution scans were performed eight weeks after discharge according to established methods.[18]

Coronary arteriography and left ventriculography[19,20] were performed on 103 patients (62 white, 41 Asian) within 12 weeks of infarction. Three months later (to ensure unfamiliarity) the angiograms were scored by two experienced observers who were unaware of the patients' names or ethnic origin. In cases of disagreement a third consultant opinion was sought from outside the department and a consensus agreed. For each patient the numbers of vessels with $\geq 50\%$ luminal stenosis seen in a minimum of two views were identified. Patients with no appreciable occlusive disease were classified as having single vessel disease and those with left main stem stenosis as having three vessel disease. In addition, vessels not associated with infarction were identified from the electrocardiogram, contrast ventriculogram, and perfusion scintigram. The number and severity of lesions in these vessels were identified by using a scoring system modified from that of Humphries *et al*[21]; all lesions causing loss of $<50\%$ luminal diameter scored 1, distal lesions causing loss of 50-89% luminal diameter scored 2, proximal lesions causing loss of 50-89% luminal diameter scored 3, and all lesions causing loss of $\geq 90\%$ luminal diameter scored 4. Proximal and distal lesions were defined as described.[22] Left ventriculography was performed in a single right anterior oblique projection. A global estimate of ventricular contraction was determined by calculating the fractional shortening of the short axis dimension as described.[23,24]

All patients were followed up for one year. Patients lost to follow up (seven white, eight Asian) were assumed to have followed a similar clinical course to the remainder of their ethnic group.

Groups were compared by Student's t test (unpaired), the Mann-Whitney U test, or the χ^2 test. Results are expressed as mean differences and 95% confidence intervals. Relative risk was calculated as described.[25]

Results

Relative rate of first infarction—At the 1981 census there were 4007 men in the borough of Harrow who were classified as Asian. They included all men in families in whom the head of the household was born in India, Pakistan, or Bangladesh. Those in families in whom the head of the household was born in east Africa were also classified as ethnic Asians because a random sample of five local general practitioner registrars had failed to identify a single black person born in east Africa and none was known to the

respective practitioners. There were 29 900 men who were classified as white. Figure 1 shows the age distribution of the two ethnic denominator populations together with the age specific rates of infarction in each ethnic group. The difference in relative rates of infarction was greatest in the youngest age group. Given that no ethnic bias in referral to hospital by general practitioners was found, the overall relative risk of infarction was estimated to be 4·9 (95% confidence interval 3·4 to 6·9) times higher in Asian than white men aged 30-59 at the 1981 census. The mean ages at presentation were 50·2 (SD 9·0) years in Asians and 55·5 (6·7) years in white patients (mean difference 5·5 years (95% confidence interval 2·5 to 7·1); p<0·0001). The mean ages of the two denominator populations in 1986 were estimated as 47·1 years for Asians and 49·5 years for white people, based on the number of people in each ethnic group in each five year age group (30-59) at the 1981 census.

Clinical history—Clinical presentation in the two groups was similar. Thirty (59%) of the Asians and 52 (68%) of the white patients were referred by general practitioners. The median time from onset of symptoms to arrival in the coronary care unit was identical in the two groups (4·0 hours, range 1-24). In both groups 16% of patients (eight Asian, 12 white) were referred more than 24 hours after the onset of symptoms. Antecedent angina was present in 12 (24%) Asians and 16 (21%) white patients. Six patients in each ethnic group (12% of Asians, 8% of white patients) were known hypertensives (difference not significant). Among smokers the mean daily cigarette consumption was 16·5 for Asians and 17·5 in white patients, but total cigarette consumption as assessed by packs a day times years was 29·3 (range 5-100) in white patients and 19·9 (range 5-60) in Asians (p<0·05). Twenty (39%) Asians were non-smokers as compared with 25 (32%) white patients. One third of patients in each ethnic group had a positive family history of ischaemic heart disease. Two features of the history were different, and these concerned diabetes and pain. Diabetes had been previously diagnosed in nine (18%) Asians but only two (3%) white patients (p<0·001). None of the Asians was taking insulin and all but two were controlled by diet alone. One white diabetic was insulin dependent. Pain was deemed atypical in 26 (51%) of the Asians and 30 (39%) of the white patients. Among men with atypical pain the Asians had a mean pain score of 2·24 (SD 1·09) and the white patients a mean pain score of 1·50 (0·62) (p<0·05).

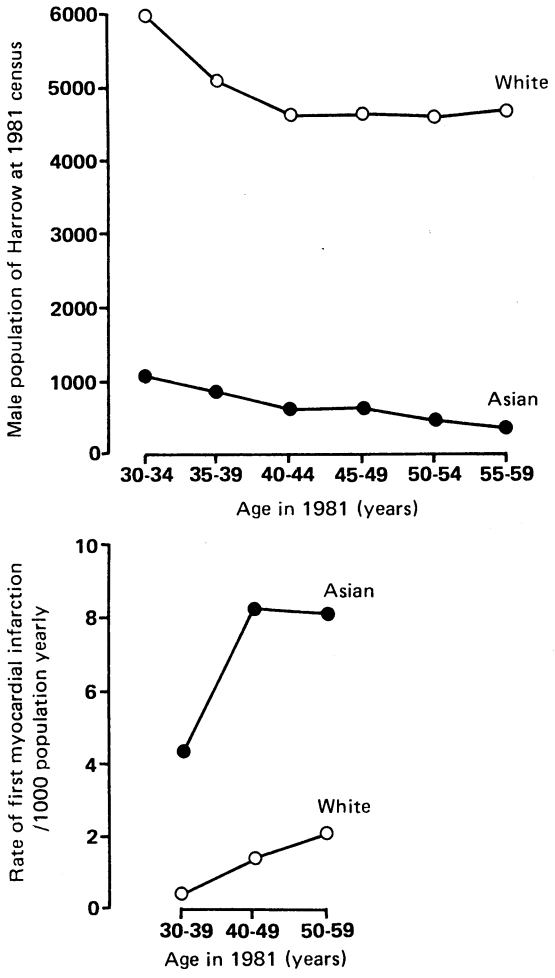

FIG 1—*Age distribution of ethnic Asian and white male populations (within range 30-59 only) living in Harrow at 1981 census and age specific rates of first myocardial infarctions based on age in 1981*

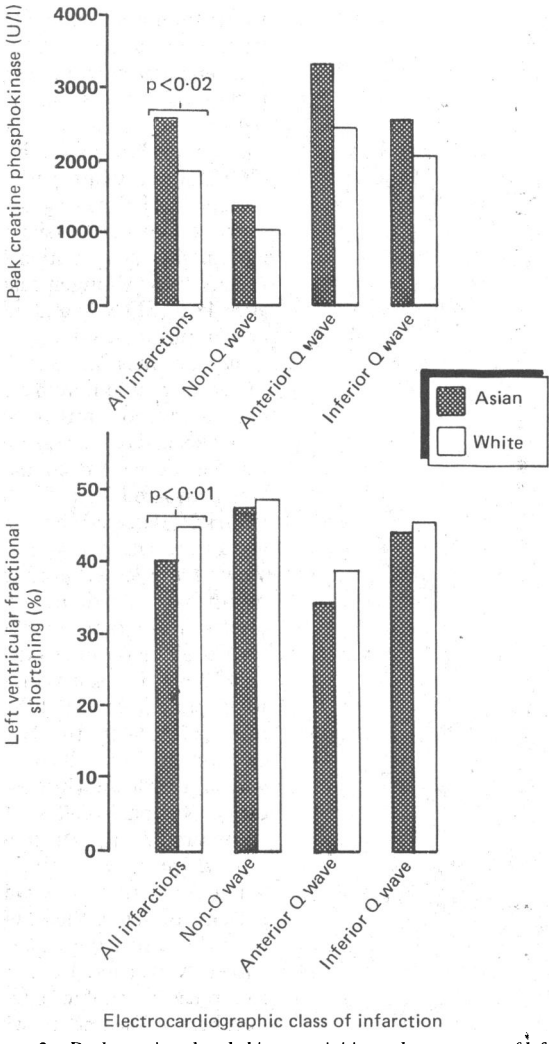

FIG 2—*Peak creatine phosphokinase activities and percentages of left ventricular fractional shortening in Asian and white patients stratified by electrocardiographic subtypes of myocardial infarction. (Note that trend established for "all infarctions" in respect of each measurement persisted in all electrocardiographic subtypes)*

Electrocardiography—Fifty one (66%) of the white patients and 38 (75%) of the Asians had Q wave infarcts. Of these, 24 (47%) and 23 (61%) respectively were classified as anterior.

Creatine phosphokinase—The mean peak creatine phosphokinase activity in Asians was 2588 (SD 1891) U/l and in white patients 1811 (1423) U/l (mean difference 777 (95% confidence interval 155 to 1399) U/l; p<0·02). In neither group was there a significant age effect on peak activity. Figure 2 shows the mean creatine phosphokinase activities stratified by the electrocardiographic subtypes of infarction in the two ethnic groups. In all subgroups the mean values were higher in Asians, and in both ethnic groups

values for non-Q-wave infarction were lower than for Q wave infarction ($p<0.01$).

Exercise electrocardiography—There were no significant differences in exercise electrocardiograms between the groups, roughly a quarter of patients in each group having angina as the end point and 20/55 (36%) of the white patients and 11/27 (41%) of the Asians studied having ST depression >2 mm. Mean exercise time in the Asians was 8·76 (SD 4·22) minutes and in the white patients 9·72 (4·52) minutes. The Asians were younger than the white patients (mean ages 48·5 (SD 8·4) and 55·4 (7·0) years respectively), and a pronounced effect of age was seen in both groups, suggesting that the observed small difference in exercise time may have underestimated the difference after accounting for age.

Radionuclide ventriculography—Resting ejection fraction derived from radionuclide ventriculography was compared with fractional shortening of the left ventricle detected by contrast ventriculography. A good correlation ($r=0.85$; $p<0.001$) was found. In view of the small number of patients in each ethnic group having radionuclide ventriculography (25) no electrocardiographic subgroup analyses were performed. The mean ejection fraction at rest was 38·8% (SD 13·6%) in Asians compared with 47·7% (14·7%) in white patients (mean difference 8·9% (95% confidence interval 1·0% to 16·9%); $p<0.05$). On exercise the ejection fraction fell to 38·0% (14·1%) in Asians and 44·2% (15·7%) in white patients (difference not significant). Regional wall scores were 3·26 (SD 1·81) in Asians and 2·21 (2·01) in white patients.

Thallium myocardial perfusion scintigraphy—Thallium scintigraphy was performed on the first 40 patients to detect those with previous silent infarction missed at presentation on electrocardiography. In no subject was a fixed defect detected remote from the electrocardiographic site of infarction. The mean numbers of segments with fixed defects were 1·93 (SD 0·62) in Asians and 1·61 (0·63) in white patients.

Coronary arteriography and left ventriculography—The mean plaque and total scores for the two observers were 2·64 (SD 1·98) and 4·41 (4·10) for observer 1 and 2·56 (1·86) and 4·46 (4·22) for observer 2; the scores recorded by observer 1 were used for further analysis. In both ethnic groups the extent of atheroma increased with age. Table I shows the proportions of patients in each ethnic group with one, two, or three vessel disease. Fewer white patients had three vessel disease ($p<0.001$) and fewer Asians had single vessel disease ($p<0.01$). The total atheroma scores for vessels not associated with infarction in patients in the three 10 year age groups (30-39, 40-49, 50-59) were: for Asians 4·94 (median 4·0, range 0-11), 6·11 (median 5·0, range 0-11), and 8·57 (median 5·5, range 1-18); and for white patients 2·63 (median 1·5, range 0-7), 2·58 (median 1·5, range 0-10), and 4·10 (median 2·5, range 0-12) ($p<0.001$, $p<0.01$, and $p<0.001$ for ages 30-39, 40-49, and 50-59 respectively). The average plaque scores were 1·68 (range 1-4) in white patients and 1·65 (range 1-4) in Asians. Fractional shortening was compared between ethnic groups in all electrocardiographic subgroups; figure 2 summarises the results. Overall fractional shortening was 40·2% (SD 9·4%) in Asians and 45·0% (7·6%) in white patients (mean difference 4·8% (95% confidence interval 1·4% to 8·2%); $p<0.01$). In both ethnic groups fractional shortening was significantly higher in patients with non-Q wave infarction. In all electrocardiographic types of infarction fractional shortening was lower in Asians than in white patients.

TABLE I—*Proportions of patients in each ethnic group with single vessel, two vessel, and three vessel coronary artery disease (defined as ≥50% luminal stenosis)*

	Single vessel disease	Two vessel disease	Three vessel disease	Total patients studied
No (%) of white patients	26 (42)	23 (37)	13 (21)	62 (100)
No (%) of Asian patients	8 (20)	11 (27)	22 (54)	41 (100)
Significance of difference	$p<0.01$		$p<0.001$	

TABLE II—*Differences between Asian patients with impaired glucose tolerance and diabetes and Asians with normal blood glucose values. Except where stated otherwise values are means (SD in parentheses)*

	Asians with impaired glucose tolerance and diabetes	Asians with normoglycaemia	Significance of difference
No (%) studied	19 (37)	32 (63)	—
Age (years)	53·2 (7·4)	48·5 (9·5)	$p<0.07$
No with atypical pain	8	16	$p<0.8$
Peak creatine phosphokinase (U/l):			
All electrocardiographic types of myocardial infarction	2499 (1354)	2637 (2152)	$p<0.8$
Non-Q wave myocardial infarction	2187 (1588)	680 (518)	$p<0.05$
Left ventricular fractional shortening (%)	41·1 (6·8)	39·9 (7·1)	$p<0.5$
Total atheroma score (units)	5·7 (3·9)	6·2 (4·9)	$p<0.8$

Clinical outcome—Of the 113 patients who were evaluated at one year, 25 of 43 Asians and 50 of 70 white patients (difference not significant) were alive and had suffered no further cardiac events. Ten patients in each group had received surgery or balloon angioplasty. There were six deaths in the Asian group and seven among the white patients. Two Asians and three white patients had suffered a further non-fatal myocardial infarction. Comparison between Asian patients with diabetes or impaired glucose tolerance and Asians who were normoglycaemic mostly showed no significant differences (table II). In particular there was no difference in the proportion of atypical presentations. The extent of atheroma in vessels not associated with infarction was similar, and size of infarction assessed by three methods failed to identify significant differences. Creatine phosphokinase activity was higher in Asian patients with non-Q wave infarction and impaired glucose tolerance than in normoglycaemic Asians.

Discussion

The principal aim of this study was prospectively to compare the relative rate and clinical course of first myocardial infarctions in Asians and white patients. Though this is a study of survivors, by examining the clinical presentation, acute progression, and post-infarction risk state of the two ethnic groups some pathophysiological factors responsible for the previously described high incidence of ischaemic heart disease in Asians might be identified. Many studies have attributed the higher levels of morbidity and mortality from ischaemic heart disease in Asians to atheroma.[10] This assumption is not unreasonable, but, though coronary atheroma acts as a substrate for infarction in most cases, other factors undoubtedly play a part.[26,27] This study was therefore also designed to test the hypothesis that the excess morbidity and mortality from ischaemic heart disease in Asians is associated with a higher incidence and greater degree of coronary atheroma. First infarction was chosen because the changes in coronary anatomy were likely to be more easily defined in patients presenting with a first myocardial infarction. Finally, the study allowed comment on the role of diabetes in the pathophysiology of infarction in Asians.

Most Asians in this study (39/51; 76%) were Gujaratis who had migrated to Britain from east Africa. The remainder were a mixture of other southern Asian cultures. Comparisons were made between all Asians

and all white patients because, though the 1981 census allows some further ethnic classification (assuming that all east Africans were Gujaratis and all Pakistanis were Moslem), a proportion of Gujaratis came directly from India and some Moslems came from east Africa. In view of this a more accurate ethnic assessment of the denominator population was considered impractical. Furthermore, other work suggests that the increased risk of ischaemic heart disease applies to many immigrant Asian groups with different cultural, religious, and socioeconomic conditions.[1 4]

Other workers have calculated the rate of infarction in Asians as between 1·4 and 5·0 times greater than in white people.[1 5] In this study only patients who were resident in the borough of Harrow at the 1981 census were included. This facilitated an estimation of the relative rates of infarction for the two ethnic groups using the 1981 census to derive the denominator populations. Differential migration from the borough of either ethnic group between the 1981 census and the period of study five years later may have given rise to some error in calculating infarction rates. Nevertheless, more than 80% of the white male population would have to have moved in order to explain the observed difference in relative infarction rate on the basis of migration alone. Such an exodus was not apparent, and the general practitioners' estimate (taken at the time of the study) of an 80% white and 20% Asian population within the borough supports this. An estimation of bias in referral patterns by general practitioners failed to identify any organisational reason for the different rates. The similar presentation time between the ethnic groups also indicated that infarction resulting in death within the first few hours was an unlikely source of bias.

Another possible source of bias concerns the number of people in each ethnic group who worked outside the hospital catchment area. This, however, would have affected at most fewer than one third of all patients. Finally, estimation of the two ethnic denominator populations by using the 1981 census may have underestimated or overestimated the number of Asians resident in Harrow at that time. Nevertheless, despite these potential sources of bias and the possible errors in estimating the denominator populations this study strongly suggests an increased rate of first myocardial infarctions in Asian men. Though the mean age of the Asian denominator population was 2·4 years less than that of the white population, the mean difference in age at first infarction (5·5 years) was greater and the difference in age specific rates of infarction was greatest in the 30-39 years age group. This suggests that first infarction occurs at a younger age in British Asians than in white people.

The size of myocardial infarction assessed by three methods (peak creatine phosphokinase activity, radionuclide ventriculography, and contrast ventriculography) was significantly greater in Asians, even when allowing for the different proportions of each electrocardiographic type between ethnic groups. The difference in creatine phosphokinase activity and residual ventricular performance was not a function of the greater extent of coronary artery disease in Asians. No significant difference in creatine phosphokinase activity or fractional shortening was found between patients with single vessel disease and those with three vessel disease. Similarly, patients subsequently found to have diabetes or impaired glucose tolerance did not have higher creatine phosphokinase activities or a lower percentage fractional shortening than normoglycaemic subjects. This implies that the larger areas of infarction in Asians were not related to a higher incidence of glucose intolerance or the greater extent of large vessel coronary artery disease.

Serum lipid concentrations were not measured at the time of acute infarction in this study. Previous workers, however, have shown that Asians have higher free fatty acid and triglyceride concentrations than white people,[28 29] and evidence suggests that high concentrations of triglycerides and free fatty acids may predispose to greater ischaemic damage in jeopardised myocardium.[30] The mechanisms by which such damage arises may be through interruption of cell membrane function, mitochondrial respiration, or intracellular enzymes.[31 32]

The pattern of coronary artery disease in Asians, particularly the greater number of lesions in vessels not associated with infarction, mirrors the findings in other patient groups with a predisposition to atherogenesis.[33 34] In patients with hyperlipidaemia not only is the proportion of subjects developing clinical disease greater but also the extent and severity of atheroma are greater than in patients with normal lipid patterns.[33] The finding of more extensive coronary atheroma in Asians therefore probably reflects a greater propensity to develop atheroma rather than a lower threshold for infarction at any given degree of coronary atheroma. The combined findings of a higher relative rate of infarction, the occurrence at a younger age, and the more extensive atheroma are highly suggestive that atherogenesis is either accelerated or premature, or both, in Asians compared with white people.

Patients were followed up for one year. Though no significant differences in the rate of further cardiac events between ethnic groups were seen, the low number of Asians free of further events, particularly surgical intervention, reflects the greater extent of coronary atheroma. Other studies have shown that long term prognosis correlates well with the extent of coronary disease.[21]

Premature or accelerated coronary atherogenesis is almost certainly a main contributory cause of the high morbidity and mortality from ischaemic heart disease in Asians. In this study, however, the proportion of Asian patients with diagnosed diabetes was not different from the prevalence of diabetes in the immigrant Asian community in north west London. Additionally, the severity of coronary atheroma was not worse in Asians with impaired glucose tolerance and diabetes compared with normoglycaemic Asians, and infarction did not occur at a younger age in diabetics. This suggests that the excess rate of myocardial infarction in Asians is not directly attributable to the concurrent finding of diabetes. The finding of larger infarctions in Asians also raises the possibility that previously described high concentrations of triglyceride in Asians may be responsible for both the excess atheroma and excess ischaemic damage.

Further examination of the relation between concentrations of triglycerides, high density lipoprotein cholesterol, glucose, and insulin in Asians might help to identify a pathophysiological mechanism (possibly related to insulin resistance) which would explain the apparently unrelated findings of high rates of coronary artery disease and diabetes in Asians.

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Optimisation of positive end expiratory pressure for maximal delivery of oxygen to tissues using oesophageal Doppler ultrasonography

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Abstract

***Objective*—To assess oesophageal Doppler ultrasonography as a convenient means of optimising positive end expiratory pressure for maximal delivery of oxygen to tissues.**

***Design*—Measurements of blood flow, arterial oxygen saturation, and cardiac output by thermodilution (when available) at baseline and at 20-30 minutes after each incremental increase (2·5-5·0 cm H_2O) in positive end expiratory pressure to a maximum of 20·0 cm H_2O. If the cardiac output fell by more than 15% measurements were repeated after stepwise decreases in positive end expiratory pressure. No other manoeuvre such as endotracheal suction or changing ventilator settings, drug or fluid dosage, or the patient's position was performed for at least one hour before the start of the study or during it.**

***Setting*—Intensive care unit.**

***Participants*—10 Patients being mechanically ventilated for acute respiratory failure who had stable haemodynamic and blood gas values and required a fractional inspired oxygen concentration of $\geq$0·45. They were assessed on a total of 11 occasions.**

***Interventions*—Incremental increases in positive end expiratory pressure followed when indicated by stepwise decreases.**

***End point*—The positive end expiratory pressure providing maximal delivery of oxygen to tissues.**

***Measurements and main results*—Arterial oxygen saturation increased with positive end expiratory pressure in all patients by an average of 6·1%. In nine of the 11 studies, however, cardiac output fell by 15% to 30% after the second increment. On the two other occasions cardiac output and oxygen delivery rose by up to 54%. Positive end expiratory pressure was decreased on seven occasions; there was considerable individual variation in the time taken for cardiac output to rise and arterial oxygen saturation to fall. In six patients good agreement was seen between the results from Doppler ultrasonography and thermodilution, the mean of the differences being −0·3% with narrow limits of agreement (−14·4% to 13·9%).**

***Conclusions*—Oesophageal Doppler ultrasonography is a rapid, safe, and reliable technique for optimising positive end expiratory pressure to obtain maximal delivery of oxygen to tissues. The results show the need to consider haemodynamic consequences when altering positive end expiratory pressure.**

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Introduction

Positive end expiratory pressure is commonly used to increase the oxygenation of blood in patients who are being mechanically ventilated. The increased oxygenation, however, has to be offset against a depression of cardiac output, which occurs at varying positive end expiratory pressures in different patients[1-8] and with time in individual patients.[7] Factors such as left ventricular preload[4 5 7] and right ventricular function[9] play a part in determining when and to what extent this decrease in output occurs. Increasing positive end expiratory pressure will therefore alter unpredictably the equation of oxygen delivery=cardiac output× arterial oxygen content. The delivery of oxygen may even fall below the starting value and pass unrecognised without the monitoring of cardiac output; the perceived improvement in the oxygenation of blood may in fact disguise an appreciable drop in the delivery of oxygen to the tissues. Indeed, criticism has recently been voiced about the current trend of increasing positive end expiratory pressure without due regard to the haemodynamic consequences.[10]

Thermodilution is the main technique used in inten-